
NORTHUMBERLAND & DURHAM
MEDICAL SOCIETY.

FEBRUARY 10, 1881.

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NORTHUMBERLAND AND DURHAM MEDICAL SOCIETY.

THE FIFTH MONTHLY MEETING was held in the Library of the Newcastle-on-Tyne Infirmary, on Thursday, February 10th, 1881, —Dr. Eastwood (the President) in the chair.

The following gentlemen were elected members of the Society:—

John F. Le Page, L.F.P.S. Glasg., L.R.C.P. Edin., Brandon.

Hugh Russel, M.D., Ferryhill.

Motherwell Duggan, M.R.C.S., Castle Eden.

The following gentlemen were proposed for election:—

Mark A. Wardle, L.R.C.P. & S. Edin., Bishop Auckland.

Robert Purdie, M.B. C.M. Edin., Gateshead.

PREVALENT DISEASES OF THE DISTRICT.

The PRESIDENT read the following letter which he had received from Arthur Pease, Esq., M.P., respecting the spread of infectious diseases through elementary day schools:—

House of Commons Library,

January 19th, 1881.

MY DEAR SIR,—I have had an opportunity to-day of seeing Mr. Mundella upon the petition of the Northumberland and Durham Medical Society, which was prepared for presentation to the President of the Local Government Board, a copy of which I received from you. Mr. Mundella intimated that the question of requiring a certain number of attendances for children in elementary schools is under consideration; and that probably alterations will be made in the code which will remove the liability to infection from the cause to which attention is directed in the petition under head (b), and proposed regulation 4. With regard to further medical inspection, he does not think it likely that any alteration will be made.—Yours very truly,

Dr. Eastwood.

ARTHUR PEASE.

On the motion of Dr. PHILIPSON, seconded by Mr. H. E. ARMSTRONG, and supported by Dr. Yeld, a hearty vote of thanks was accorded to Mr. Pease for the interest he had taken in the matter.

The President undertook to convey to Mr. Pease the vote of thanks.

Mr. HENRY E. ARMSTRONG presented the following:—

Return of Admissions to and Death at the Newcastle Fever Hospital during the Month of January, 1881.

	Cases.	Deaths.
Typhus	3	1
Enteric Fever	4	0
Scarlet Fever	1	0
	—	—
Total	8	1

The cases of typhus were associated, two being brother and sister, and the third their neighbour. The fatal case terminated on the 12th day, death being due to asthenia. Three of the cases of enteric fever were from the same family.

PATHOLOGICAL TRAY.

Dr. ARNISON showed a gall bladder, filled with gall stones, and the common duct blocked. The patient, a woman 43 years of age, had previously suffered from attacks of gall stones, but her death arose from peritonitis, without any apparent cause.

Dr. PHILIPSON presented the lungs and intestines from a case of general tuberculosis. The patient, an Orkney sailor, aged 25, had been three months under his care in the Newcastle-upon-Tyne Infirmary. At the time of his admission, he was suffering from chronic diarrhœa, with abdominal distention and tenderness. He also complained of cough, loss of appetite, and debility. He stated that he had lost weight. The temperature, morning and evening, was 100° Fah. Upon physical examination of the chest, the movement was found to be impaired, the percussion resonance was diminished in the infra-clavicular regions, and the respiratory and vocal sounds and resonance were exaggerated. There were no moist rhonchi. Associating the abdominal condition with the chest manifestations and the persistent exaltation of temperature, the case was regarded as one of general tuberculosis and not enteric fever. For a time he improved somewhat. Subsequently, it was noted that as the intestinal irritation subsided, his cough increased, and instead of continuing dry, he began to expectorate much purulent matter, and that the physical signs indicated progressive action in the lungs. Gradually he became weaker, and for the last few days of his life, lay in a state of semi-stupor. At the autopsy, the lungs, when cut into, exhibited throughout small nodules of tubercular deposit, vomicœ being present in both upper lobes. The peritoneum was here and there studded with miliary grey tubercle, more especially over the small intestines. The mesenteric glands were much enlarged, and when cut into were found to consist of yellow tubercle. Upon laying open the small intestines, at the lower end of the ilium, small round deposits of tubercle were met with, corresponding to the solitary glands, also large deposits in the peyerian patches. Near the lower end of the ilium, also at the commencement of the large intestine, ulcers, round and oval, running transversely across and around the intestinal canal, were seen. The chief interest of the specimens was the illustration of catarrhal phthisis of the lungs associated with tubercle of the intestines, peritoneum, and mesenteric glands. The case was further of interest, in a diagnostic point of view, as exemplifying the

difficulty of distinguishing general tuberculosis from enteric fever ; and also, as to its course, as exemplifying the proposition, that when the intestinal irritation is relieved, the disease in the lungs takes on an aggravated action, and *vice versa*.

Dr. PHILIPSON exhibited the heart, kidneys, and spleen from a case of chronic albuminuria. The patient, a single woman, general servant, aged 24, had been under his care, in the Newcastle-upon-Tyne Infirmary, for two months anterior to her death. At the time of her admission, she was suffering from general dropsy (internal and external); the urine was loaded with albumin, waxy casts of the tubules being easily recognised under the microscope, she complained of imperfection in her vision. Upon ophthalmoscopic examination, the optic discs were found to be milky, the retinæ being studded with minute hæmorrhagic spots. The heart was hypertrophied, more especially the left ventricle, and there was a low-pitched mitral systolic murmur. At the autopsy the kidneys were found to be examples of the large white variety of tubular degeneration. In both there were several old infarctions. The spleen was enlarged, very firm, and was the seat of several old infarctions. The heart was greatly enlarged, the walls, more especially those of the left ventricle, being greatly hypertrophied. The mitral valve was thickened. On each curtain, also, in the left auricle, were masses of adherent decolorized fibrine. The brain was very firm and dry, the optic nerves were also condensed, and minute hæmorrhages were distinctly recognisable. The case was of interest as illustrating the consequential effects of chronic albuminuria upon the organs, principally those of the circulatory and nervous systems.

Mr. HENRY E. ARMSTRONG said he thought the specimen of phthisical ulceration of the intestine exhibited by Dr. Philipson exceedingly interesting. The question of the differential diagnosis between tubercle of the intestine and some cases of typhoid fever was by no means an easy one.

Dr. HEATH showed a hydatid cyst removed from the arm of a man 40 years of age, and said:—Mr. President and gentlemen,—The members of the Society will observe that the cyst is split open and inverted ; a smaller cyst within the large one is in the same condition. The cysts are hardened at present, having been in spirit, but the thickness of the walls is still apparent. The cysts are ovoid in shape, with a prolonged and somewhat narrow neck ; but there is no head, and no hooklets were found. Before removal, the tumour, which was deep-seated in the arm, having the brachialis muscle spread out over it, as well as the great nerves and brachial artery, presented an indistinct fluctuation, and resembled a cystic sarcoma. The operation was not attended by any difficulty. The

biceps muscle was turned aside, and the brachialis, thinned out by expansion, divided in the direction of its fibres. A bag of condensed connective tissue loosely surrounded the hydatid cyst; and at the moment of dividing this bag the hydatid cyst split—the knife not having touched it—but, as it seemed, in consequence of a contraction of its walls. After the removal of the large cyst the smaller was found within, and also split in precisely the same manner as the large one. The small cyst could not possibly have been touched by the knife. Both cysts also inverted themselves on removal. The patient made a good recovery, the wound being healed in about 14 days. The cysts probably belong to the acephalocyst variety, although these are not so often found in connection with muscle as in other situations; whilst the cysticercus is more frequently met with in muscular tissue. The shape, too, is less globose than the usual acephalocyst, still I incline to think that the preparation before us belongs to that class.

Dr. HEATH, in reply to Dr. Gibson, said no hooklets had been found in the contents of the cyst.

Dr. DRINKWATER said: Mr. President and Gentlemen,—This heart I obtained from a woman, aged 49, who died on the 7th ultimo, from pneumonia and pleurisy, with effusion on the left side. It is in an advanced stage of fatty degeneration and fatty deposition. There is a considerable quantity of fat deposited on the *exterior*, principally in the anterior part of the auriculo-ventricular groove; but also extending in a thinner layer over the ventricles. The muscular substance in all parts of the organ is soft and pale from fatty degeneration. In the interior of the heart there are, in addition to a large deposit of fat in the auricular appendix of the right auricle, three large polypi. One is attached to the chordæ tendiniæ, near their point of union with the flaps of the tricuspid valve; it extends three or four inches along the pulmonary artery, becoming very narrow opposite the commencement of that vessel; a second, and similar one, springs from the mitral valve, and passes along the aorta. The third is confined to the left auricle, being broad and flattened, and having three attachments—one to the roof of the auricle, another to the left side near the auriculo-ventricular surface, and a third to the valve itself. They are all composed of fat, and have no deposit of fibrine upon them.

Mr. PAGE said: This specimen of stricture of the rectum, sir, was removed by the pathologist, Dr. Drummond, from the body of a woman, aged 46 years, who died under my care in the Infirmary a few days ago. The disease was of six years standing, and during that time, on more than one occasion, an abscess formed near the anus. The contraction is situated about four inches from the anus, and seems to be caused by a thickening of the sub-mucous

cellular tissue, analagous to simple stricture of the œsophagus. Below the stricture the gut was contracted, and its sides matted together by not very firm adhesions, apparently the result of inflammatory action.

Dr. DRUMMOND showed the heart, aorta (with aneurism), and lungs from a case of aneurism of the thoracic arch, which caused death by rapid asphyxia without rupture, and said: These specimens, Mr. President, were taken from the body of a young man, aged 25, a timekeeper, who was admitted into the Newcastle Infirmary three weeks previously, complaining of cough and dyspnœa, symptoms, which he thought were due to bronchitis, from an attack of which affection he had been suffering for upwards of four weeks. Two or three days after admission, on suddenly rising from his bed, he was observed to fall backwards, whilst his breathing became obstructed. Mr. Goyder, the Senior House Surgeon, on being sent for, finding the patient almost asphyxiated, and supposing the obstruction to be in the larynx, performed laryngotomy, other remedies having proved ineffectual; however, the operation was not followed by relief. Whilst matters were at this stage, in response to a summons from Mr. Goyder, I arrived, and found the patient unconscious, bathed in perspiration, and breathing with the greatest difficulty. The opening in the cricothyroid membrane was ample; it was therefore apparent that the obstruction lay below. Fearing lest it might be due to a foreign body impacted in the trachea, Mr. Goyder prolonged the incision downwards. A tube was then put down the windpipe to the obstruction, which appeared to be due to some body pressing on the trachea, and flattening it from before backwards. It was noticed that the trachea pulsated violently. The only view tenable under the circumstances to explain the attack was, that the patient was the subject of an aneurism which was pressing on the trachea. Having arrived at this opinion, we administered, by inhalation, some nitrite of amyl, with the object of lessening blood-pressure, and consequently relieving the tension of the sac. For some minutes this treatment promised favourably, so much so, that the amyl was repeated with again a slight improvement in the breathing; but the difficulty returned, and with it, apparently, the extremest exhaustion, and the patient died, practically strangled, about two hours after the first symptoms of obstructed breathing. The autopsy revealed, as we supposed, an aneurism of the transverse portion of the arch about the size of a medium-sized orange, exercising pressure upon the trachea just above the bifurcation. The sac was partially filled with laminated clot, and was distinctly sacculated. There was no rupture of the sac. The heart was normal, the left ventricle firmly contracted, whilst the right ventricle was also nearly empty, though flaccid. The lungs were congested.

Dr. Drummond said he would be glad to hear if any member of the Society had met with any cases of the sort, for he thought it was an exceedingly rare mode of death in aneurism, *i.e.*, direct pressure upon the trachea, causing death rapidly through asphyxia, without rupture.

Dr. GOWANS asked if Dr. Drummond had endeavoured to explain the sudden attack which resulted in the death of the patient? He could understand spasm of the glottis from pressure on the recurrent laryngeal causing death in cases of aneurism. Also, had the sign in the trachea, which Dr. Drummond described at a previous meeting as occurring in thoracic aneurism, been observed in the present case?

Dr. DRUMMOND said he could only imagine that the aneurism had undergone a very rapid enlargement in the direction of the trachea, perhaps due to the sudden change in the patient's posture, causing increased blood pressure. He knew that the late Professor Stokes used to teach that aneurisms did suddenly enlarge in new directions, giving rise to the supposition—when the enlargement was away from the chest wall—that sudden cure had taken place. As regards the tracheal sign, it had not been sought for; only a very cursory examination of the patient had been made in the out-patient department during the reception, and after the sudden attack the stridor was so loud that nothing satisfactory could be arrived at.

Mr. PAGE introduced a patient suffering from a chronic disease of the tongue, and said :—Twenty years ago, sir, this man, who is a pitman, and 60 years of age, noticed in the centre of the dorsum of the tongue a small growth. Slowly but steadily this growth spread until, as you will see, it now occupies the greater part of the anterior half of the tongue superiorly. Its appearance is peculiar. It is raised, and seems to consist of the papillæ of the tongue enlarged and elongated. It is hard to the touch. There is no ulceration of its surface, nor any glandular enlargement in its neighbourhood. The patient has no pain, and only complains of inconvenience in speaking. He masticates and swallows readily and easily. Three months ago, an abscess formed in the substance of the tongue; was opened, and has healed. The man has been a great smoker. He has never had syphilis, and there is no family history of cancer. He has been under my care in the Infirmary for a month, and is taking 40 grains of iodide of potassium three times a day, with three grains of hydrargyrum cum cretâ at night, and using a bichloride of mercury wash. No caustic has at present been applied. The growth is less than it was. The question is, what is the nature of the disease, and upon this point I am more particularly anxious to elicit the opinion of the members of this Society.

ON PLEURITIC EFFUSIONS.

BY CHARLES GIBSON, M.D.

Notwithstanding the achievements of practical medicine in modern times, and the extraordinary accuracy which physical diagnosis has attained to in diseases of the chest, the occurrence of inflammatory disease in this region of the body, as made manifest by *post-mortem* examination only, is not infrequent. It is in the experience of every one, much accustomed to make *post-mortem* examinations, to find, for example, the effects of acute inflammation of the pleura in the form of even extensive adhesions, when no suspicion had existed of the presence of acute pleuritis during life. The same thing is observed, from time to time, in the matter of pleuritic effusions.

Again, the presence of mere watery fluid in the cavity of the pleura—Hydrothorax—the dropsy of obstructive disease—must not here, or indeed, in any way, be confounded with the fibrino-serous effusion of pleuritic inflammation, nor can it be admitted that there is ever any intimate connection between these two affections.

But the manifestation of an empyema constantly bears close relationship with that of fibrino-serous effusion. Neither the purulent nor the fibrino-serous accumulation can possibly occur without pleuritis; except, perhaps, in those very rare cases where abscess of the thoracic walls bursts into a pleural cavity. Fibrino-serous effusion is the almost constant primary outcome of pleuritic inflammation. Purulent accumulations are almost invariably developed upon the fibrino-serous, and puncture of the pleural wall for the removal of fibrino-serous effusion has been speedily followed by the appearance of pus in the fluid still retained in the thoracic cavity, and in that which has been subsequently effused. Here it is assumable that one of two things has taken place: either white blood cells, which have passed from the pleural membrane, have undergone transformation into pus cells, and mingled and multiplied in the fibrino-serous fluid; or the membrane itself, injured by the operation of puncturing, has undergone a still more pronounced inflammation than held before; blood corpuscles have been transuded from inflamed vessels; these have undergone progressive changes—proliferations; pus cells have been developed in the pleural membrane itself, and thrown off as such into the pleural cavity. Perhaps both conditions may hold, either together or separately. At least this much is certain: that when the pleural cavity has been occupied by a large quantity of purulent matter, the result of pleuritis, the pleural membrane has become greatly changed in morphological constitution, in density,

and in thickness—has become so greatly changed, that probably no well-authenticated case of empyema has ever been reported in which the pleural membrane has been restored to normal, or nearly normal, conditions. If this is true, then it follows, logically, that *lung textures*, which have been bound down by pleura, so greatly changed by inflammatory processes, are never restored to their normal condition by time, nor by any medical treatment whatever; and that the space left after the removal of the purulent matter by the operation of paracentesis is only obliterated by the contraction of chest walls and the displacement of thoracic organs. Such a cavity, until its obliteration has been effected, is occupied by air, with or without cheese-like remains of inspissated pus.

Untouched fibrino-serous effusions sometimes remain for long periods without perceptible change. It may safely be accepted, however, that these accumulations are not always of so innocent a character. Even purulent accumulations occasionally appear to be triflingly irritant, but the presence of pus in a pleural cavity is, like the presence of ichor, for the most part highly irritating. The pleural membranes, by virtue of their thickened condition in empyema, doubtless have become more resistant; but the irritant quality of the pus is still operative. Erosion takes place. The subserous connective tissue becomes pressed upon directly. Bulging at some point of the thoracic walls follows. The common tegument becomes necrosed, and the pus flows from the cavity. In the curative process sometimes set up in fibrino-serous effusions, the exuded fluid becomes gradually more and more dense, and smaller and smaller in quantity, until the dispersion of the fluid. Occasionally, however, as in the case of empyema, a caseous yellow residue is left behind, which is constituted of fibrinous and cellular matters. Even here, if much false membrane has been formed upon the inflamed pleura, the lung beneath it may never expand, and be useful as before, for the purposes of respiration. But in the fibrino-serous form of the pleuritic disease the amount of interstitial and superficial fibrinous deposit is commonly small as compared with that of empyema; and this textural condition is usually more susceptible of degenerative processes and of dispersion. The pleural membrane in empyema is invariably dense and thick, and the possibilities of its dispersion are scarcely calculable. Lastly, it may reasonably be expected that compressed organs and parts will attain to liberty of action, from the contractile grasp of the diseased membrane, in fibrino-serous pleuritis, under favourable circumstances. But such expectations are not justifiable in any case of empyema.

The treatment of these two forms of pleuritic effusion is essentially different. It has varied very much from time to time. Successive waves of knowledge in this regard have altered the pur-

pose and the agency of the physician. The physical diagnosis, as set forth by Lænnec and perfected by his followers, has done very much for the subject of thoracic disease, and the employment of the exploratory trochar in our day has certified our physical diagnosis in cases of pleuritic effusions. Nevertheless, their fatality has hitherto been very large. Our own Addison protested loudly and persistently against paracentesis in these affections, believing it to be chargeable with much of their fatality. And many others have entertained an equally unfavourable view of the operation. In a vast number of cases of pleuritic effusion the agency of drugs has been utterly futile. It is now recognisable that much of the want of success in the management of pleuritic effusions in times past is traceable to the employment of identical treatment in essentially distinct forms of disease. It may safely be asserted that no medicinal agent known has the least curative action on empyema. Its surgical treatment, however, is very commonly successful. In fibrino-serous affections, on the other hand, the operation of paracentesis often fails and often is injurious. Happily, in these affections medicinal agents are often curative. To the influence of antiseptics during and subsequent to the operation of paracentesis in empyema, supplemented by the employment of the drainage tube, is doubtless due much of the marvellous success which now holds in the treatment of this hitherto commonly fatal disease. The results of this treatment in cases under my own care in this Infirmary and elsewhere, and in that of others, have been almost everything which could reasonably have been hoped for. The treatment of empyema, then, is exclusively surgical, and the employment of the trochar, the knife, and the drainage tube, under antiseptic precautions, is carried out with exceedingly little risk. In fibrino-serous effusion, both medical and surgical means have been successful from time to time: And the employment of paracentesis sometimes becomes a matter of urgent necessity in consequence of the severe pressure which the fluid contained in the pleural sac produces upon the circumjacent parts, and the removal of the fibrino-serum by surgical operation has in itself been sometimes followed by a complete cure of the disease, just as has happened from time to time in ascites, &c. The operation has commonly failed, however, and not unfrequently it has added largely to the dangers of the disease. In certain constitutional states, again—cachexias—no medicinal treatment whatever is competent, probably, to effect the restoration of the sufferer to health. Nevertheless, in cases of fibrino-serous pleuritic effusions, unassociated with cachectic conditions, something very like *specific action* is exerted by *mercury*. Doubtless mercury has for ages been employed in the treatment of pleurisy. It has also been employed therapeutically in pleuritic effusions. It has, however, been

employed somewhat loosely in these maladies commonly, and the results of such employment, as might be expected, have often been unsatisfactory. Mercury is positively injurious in empyema; but in suitable cases of fibrino-serous effusion it exercises a remarkably salutary influence. This it does in two ways. In the first place, it determines the absorption of the fluid from the pleural cavity, and in the second place, it alters the solidified fibrinous deposits upon and within the affected membrane in such wise that they are removed by the absorbents. The compressed lung tissue thence is enabled to expand; normal respiration follows.

The agency of antiphlogistics, such as local bleeding, counter-irritants, and sedatives, is rarely useful, and only applicable in the earlier stage of the diseased action; while in the established conditions of the disease, diuretics, purgatives, and sudorifics—commonly had recourse to—cannot be reckoned as valuable curative means. Possibly iodide of potassium, in its kinship with, and its capability as an aid to, mercury, where the action of the latter has been interfered with by peculiar conditions on the part of the patient, may prove valuable. However this may be, *the special remedy* for fibrino-serous pleuritic effusions is still *mercury*. Here are examples of its curative action—others could easily be added:—A man was received into the Newcastle Infirmary, under my care, on the 22nd day of April, 1879, with well-marked symptoms of effusion into the left pleural cavity, the result of an attack of pleurisy. The special treatment was adopted without delay, although the patient had suffered slightly from dyspnoea, the effect of pressure upon the organs of respiration and circulation. Five grains of pil. hydrarg. and one-third of a grain of opium were given to him four times a day, and a simple, unirritating, but nutritious diet was adopted. This treatment was steadily pursued until the 13th day of May—21 days. During the last few days of this period slight ptyalism was present. On the 13th day of May, careful examination showed that the chest was quite free from pleuritic effusion, and that the respiratory function of the affected lung was perfect. A second almost identical case occurred under my observation, and was again the evident consequence of inflammation of the pleura. Before the case came under my observation the operation of paracentesis had been performed, and gave great immediate relief to the patient. The fluid, however, re-accumulated. This patient, as in the case just briefly referred to, was placed under the influence of mercury, and as ptyalism was being manifested, the effusion almost suddenly disappeared, and the function of the respiratory organs again assumed its normal condition.

NOTES OF A CASE OF OVARIOTOMY.

By T. W. BARRON, M.D., Surgeon to the Durham County Hospital.

Margt. L., aged 34 years, a married woman with three children, the youngest four years of age, was admitted into the Durham County Hospital about the end of December of last year, suffering from a very large abdominal tumour, which, on examination, was diagnosed to be a tumour of the right ovary. She had first noticed the swelling, chiefly to the right side, about twelve months before admission to the hospital, and there was a history of pain in the same side prior to the discovery of the swelling. There had never been any interference with the catamenia. She had lost a great deal of flesh, looked anxious, jaded, and careworn; and looked ten or fifteen years older than she really was. The tumour had never been tapped. There was no interference with the action of the bowels or bladder; and on examination with the sound the womb was found to be free, and of natural size and condition. The case was considered a favourable one for operation. Accordingly, on Thursday, January 6th, with the kind assistance of Drs. Dixon, Mason, and Oliver, ovariectomy was performed. The operation was done with full antiseptic precautions. Chloroform was the anæsthetic used. The bladder having been emptied, a small incision (about four inches long) was made. This it was afterwards necessary to slightly enlarge. The cyst having been exposed, it was tapped with the large trocar and canula, with vulsellum forceps, as recommended by Mr. Bryant. More than three gallons of a clear brownish fluid were removed, and the cyst nearly emptied. The cyst was then drawn out as far as possible through the wound with the aid of two pairs of cyst forceps. A few adhesions were found; one considerable adhesion to the omentum was ligatured in three or four portions with carbolized catgut, and cut through with the scissors. The abdominal wound had at this point to be slightly enlarged to admit of the extraction of a solid mass which formed the base of the cyst. The pedicle was divided by the finger into two portions, which were ligatured with stout carbolized whipcord. The two portions of the pedicle were then cut through with the scissors and the tumour removed, the ligatured pedicle being returned into the abdominal cavity. The bowels were never seen during the operation. There was no abdominal bleeding, only a little oozing from the cut edges of the wound. The wound was closed with six sutures, a drainage tube being left in the wound to allow the escape of any blood that might escape into the cavity. The wound was then dressed antiseptically. Patient passed a quiet day, suffering from slight chloroform sickness. Evening temp 100·6°

Friday, Jan. 7.—Morning temperature, 98·8°; wound dressed; tube removed; very slight discharge; no sickness. Evening temperature, 99·6°.

Saturday, Jan. 8.—Temperature, 99·2. Had a quiet night. Wound dressed; no discharge; no sickness. Patient is being fed by enemata.

Sunday, Jan. 9.—Wound not dressed. Patient has taken food by the mouth. No sickness. Has slept well. No pain. Feels very comfortable.

Monday, Jan. 10.—Wound dressed; no discharge.

Tuesday, Jan. 11.—Not dressed. Bowels moved with enema. Slept well. No pain or discomfort.

Thursday, Jan. 13 (7 days).—Wound dressed; three stitches removed; no discharge. Wound quite close.

Sunday, Jan. 16.—Remainder of stitches removed. Wound quite closed; dressed with boracic ointment.

Monday, Jan. 17 (11 days).—Patient up for a time, and moved into a larger ward.

Patient continued quite well, improving daily in appearance, gaining flesh and spirits, until Saturday, February 6, when she was discharged cured.

ON ANÆSTHESIA.

By DAVID DRUMMOND, M.A., M.D., Physician to the Newcastle-upon-Tyne Infirmary.

[CONTINUED FROM PAGE 104.]

In reviewing the points which are of diagnostic value in distinguishing between functional and organic sensory impairment, too much stress must not be laid upon the erratical character of the phenomena observed, as described above; for, on the one hand, in some of my cases of old standing functional disorder, not only is the anæsthesia persistent, but the phenomena are as well immutable, and, indeed, practically not to be distinguished from those of organic origin; whilst on the other hand, in some undoubted organic lesions, as, for example, in locomotor ataxy, the phenomena vary from day to day, though more in degree than kind.

As regards the obstinacy and invariableness of the phenomena in hysterical cases, my meaning may be well instanced by the following case:—

J. F., æt. 22, a young woman of no employment, living with her friends, was treated for over two years—seen occasionally—for symptoms which may be said to make up the symptom picture of so-called spinal irritation, viz., backache (“tender spine,”) headache, vomiting, inframammary pain, areas of hyperæsthesia, &c. Subsequently, she became lame on the right leg, and though regarding the halt as hysterical lameness, yet as there was pain in the hip and knee, I asked my colleague, Dr. G. H. Hume, to examine the case, lest hip joint disease might have been overlooked. Dr. Hume however, agreed with me as to the origin of the lameness. The difficulty in walking increased, and for a few weeks the patient was lost sight of. When again seen, she was the subject of nearly complete paraplegia. The paralysis was both sensory and motor; the only movement she could perform with the lower extremities was in the case of the toes of the left foot, which could be feebly flexed and extended. The motor paralysis of the right lower extremity was complete.

As regards sensation, which interests us more especially at present, analgesia of the skin was the most prominent feature—for the loss of painful impressions was most complete—though tactile impressions were only appreciated when stimuli were very severe; for example, a pin thrust between the skin and the underlying tissue for half an inch was only felt as a touch sensation, as was also a very firm grasp of the foot or leg. Thermal impressions were lost to much the same degree as tactile. Tickling was not felt. This

almost complete anæsthesia affected only the lower extremities, or to be more accurate, extended on the right side, from the crest of the ilium downwards; and on the left, the upper limit was five inches above the patella. But the anæsthesia was not strictly paraplegic, for the whole of the right side was more or less affected, except the face and neck; though to a much less extent than the leg. The trunk, upper extremity, face, and neck were not at all affected on the left side. The sensory condition, as described above, was observed about a year and a half ago, and, beyond an occasional variation in the condition of the upper part of the right side, *i.e.*, above the crest of the ilium, the phenomena have remained the same. Latterly the sensory impairment of the trunk has vanished, leaving the case one of paraplegic anæsthesia, properly so called. In this case, without entering into the features which stamp it as belonging to the functional neuroses, it may be said that the condition of paraplegic sensory paralysis existed for over eighteen months. During this time the phenomena did not vary. As there are some features of interest in the details of the sensory impairment of the case, I may be allowed to notice them briefly.

No form of stimulus applied to the skin, no matter how intense, produced pain. A strong Faradic current affecting the muscles caused great suffering. Thermal impressions were only recognised in the direction of heat; ice applied to the skin was felt as a touch sensation. It required the application of a high temperature before *warmth* was felt, *i.e.*, a metal spoon dipped in almost boiling water. In applying thermal stimuli the skin was accidentally blistered—cuticle removed; it was then observed that on the application to the blistered surface of the same stimulus which the patient before described as warm, that no sensation was felt. Also the prick of a pin, which was felt in the surrounding regions as a touch sensation, when applied to the blistered part caused no sensation. In about ten minutes' time from the occurrence of the blister, the surface—size of a sixpence—began to recover, a prick causing a very feeble sensation in comparison with the surrounding skin; but in three-quarters of an hour the blistered surface had so far recovered, that stimuli applied to the surface (denuded of the cuticle) and to the neighbouring skin, were felt alike. Then it was observed that a sharp prick into the centre of the blistered area appeared to *exhaust* the sensory apparatus of the part, for stimuli applied immediately afterwards were not distinguished. Again this *exhaustion* through stimulation was observed, by inserting a pin, and keeping up considerable pressure upon the head, so that the stimulus was continuous, if not progressive in intensity, when the sensation caused by the initial prick was soon lost, the subsequent stimulus producing no sensation. This susceptibility

to *exhaustion* on the part of the peripheral sensory apparatus I had noticed before, especially in cases of *Tabes dorsalis*, and shall have occasion to refer to the phenomenon again.

My observations have led me to believe that subjective symptoms are generally absent, or but slightly marked, in cases of hysterical anæsthesia, and when present are rather of the negative description, *i.e.*, dependent upon the diminution of normal sensation; for example, in the case just cited, the patient said she felt "as if she had no legs," it being comparatively rare to find the more positively subjective symptoms in the shape of paræsthesiæ, the "furry feeling," "formication," &c., in hysteria. This might be expected *à priori*, assuming that the paræsthesiæ are caused by disturbances (irritation) induced by the organic change in the cord or brain, and may be viewed as analogous to the motor phenomena of "twitching" of the muscles in cord and central lesions. Indeed, it is probably a mistake to associate subjective sensory symptoms with anæsthesia, considering the frequency of the occurrence of motor paralysis with subjective sensory phenomena, without sensory impairment. The frequency of this association alone, *i.e.*, motor paralysis and sensory subjective symptoms, goes far, I think, to prove in opposition to the opinion expressed by Schiff and others—from direct experiment—that the grey matter in the cord not only conducts sensory impulses, but is also excitable under direct irritation; it being generally allowed that the posterior white columns are not æsthesodic, *i.e.*, they both conduct and are susceptible to direct irritation.

It is probable that other factors besides time have influence in causing the phenomena to be persistent. I think that in cases of hysteria, where motor paralysis is associated with the sensory impairment, the anæsthesia is usually more obstinate; or rather, where sensory impairment is associated with any motor phenomena, *e.g.*, spasm, convulsive seizures, or paralysis, it is much less variable; motor phenomena being the expression of a more profound functional disturbance than sensory.

Before proceeding to the consideration of the various anæsthesiæ depending upon organic cord and brain lesions, it will be convenient to consider sensory impairment due to toxic influences. It will, I think, be conceded that the most frequent cause of toxic anæsthesia is chronic lead-poisoning. Anæsthesia in plumbism is, according to my experience, an exceedingly common symptom; indeed, it is comparatively rare to meet with a case of chronic lead-poisoning without some degree of sensory impairment. I am aware that this is not the experience of some of the principal writers on the subject. Tanquerel has only met with anæsthesia eleven times out of the large number of thirteen hundred lead cases, excluding amaurosis. Naunyn, the writer of the article on lead in

Ziemssen's *Cyclopædia*, remarks that anæsthesia is rare in lead-poisoning. It is true that the majority of my cases of lead-poisoning have occurred in young women, workers in white lead in the factories of the district, and considering the difficulty in distinguishing some cases of hysterical anæsthesia from the milder forms of lead sensory impairment, it may be that several of my lead cases have been the subject of hysteria. Whilst admitting the occurrence of mild and transient attacks of anæsthesia in chronic lead-poisoning, I cannot subscribe to the opinion advanced by Naunyn in the article in Ziemssen's *Cyclopædia* already referred to, which is as follows:—"Usually, there is anæsthesia of the skin only, rarely of the skin and deeper tissues. It has no definite relation to the muscular paralysis; it may appear with paralysis and also with colic and arthralgia. There may be both arthralgia and anæsthesia of the skin in the same locality. On the whole, in localization, and in its course it is very varying, and, as it were, freakish. The attack in one place disappears to-day only to appear in another in a few days. . . . It rarely lasts longer than fourteen days." I am fully of opinion with this author that the anæsthesia has no definite relation to muscular paralysis, but I think he is in error concerning the extent and degree of persistency of the sensory impairment. Of Tanquerel's eleven cases, in four were the superficial and deep structures affected, and seven the superficial alone. Tanquerel only once saw anæsthesia (cutaneous) with amaurosis. In all the cases which have come under my notice of lead-poisoning with opthalmic change, neuritic, or post-neuritic, there was sensory impairment. The peculiarity of the anæsthesia in chronic lead-poisoning is, I think, the frequency with which the cutaneous anæsthesia and analgesia—it is generally painful impressions which are lost—and analgesia of the deeper structures are co-existent and co-extensive.

The following case may be adduced as an example of lead anæsthesia:—

Margaret K., aged 20, a worker in white lead, which occupation she had pursued for upwards of seven years, was admitted into the Newcastle Infirmary for lead amaurosis and headache. Briefly, her history is as follows:—Three months before admission she was laid up with headache and colic; the attack had been ushered in by a rigor. About a month subsequently, the headache still continuing severe, she was seized with epileptic fits of the usual saturnine variety. Three severe fits occurred within a period of about 24 hours. Next day her sight was dim, and within a week she was quite blind. There was no recurrence of the fits. On admission it was observed that slight general motor weakness existed—especially were the extensors affected. Both discs were markedly atrophied, the pupils widely dilated, the eyeballs were

the subject of a peculiar nystagmus, the movements being of a semi-rotatory character, or, more correctly vertical, though Romberg asserts that such a vibration never occurs. The blue line on the gums was present, but indistinctly marked. The usual tests applied to the skin discovered an almost general analgesia, with but slightly impaired tactile sensibility. The face was not so much affected as the rest of the body. Whilst painful impressions were nowhere felt with normal acuteness, excepting perhaps the face, yet limited areas were scattered about, in which tactile, thermal, and painful sensibility—the latter superficial and deep—was almost entirely abolished. These areas of exaggerated impaired sensation were mostly upon the lower extremities, the outer surface of the thighs in particular, and varied in extent from minute patches smaller than a shilling to a surface which could be covered by the palm of the hand. It was found impossible to excite pain in these areas by pricking, burning, or powerful Faradic currents, even when applied with moist rheophores. A very hot piece of metal was described as cold. In this case of lead anæsthesia the condition was that of general sensory impairment, superficial and deep, but more especially in the direction of analgesia, with areas dispersed over the body, in which the impairment was very marked, affecting tactile, thermal, and painful sensations. The anæsthesia has persisted for at least ten weeks with but very slight variation, and is at present—the case being still under observation—as marked as ever. It is true that in many lead cases the anæsthesia is more limited in its distribution, and in some the impairment is confined to the skin alone. Especially have I noticed this to be the case where the complaint was simply lead colic, the sensibility of the muscles being more frequently affected in cases of saturnine arthralgia, or, more correctly speaking, myalgia. But the most profound anæsthesia is to be found in cases of encephalopathy, a condition which very often includes the less severe phenomena of colic and myalgia.

Alcohol is another recognised cause of anæsthesia. Two varieties of sensory impairment are to be met with in chronic alcoholism: first, Alcoholic hemianæsthesia—generally associated with motor paralysis of the same side—which resembles closely Hysterical hemianæsthesia, in which, according to Magnan, who ascribes the symptoms to central disease in the neighbourhood of the optic thalamus, there is more or less complete loss of sensation of one-half of the body, not confined to the skin, but the mucous membranes are affected, and also the muscles. Debove and others have observed such cases. Again, there occurs a general impairment of sensibility in chronic toppers, painful impressions being especially lost all over the body. This

latter form is much more obstinate than the hemianæsthesia. In a case recorded by Debove, a weak continuous current dispelled the hemianæsthesia in 30 minutes without any transference phenomena.

Another cause of anæsthesia I would mention in this connection is bromide of potassium. I have frequently induced a tolerably high degree of analgesia in patients by the use of this salt; its action upon the mucous membrane of the soft palate and throat is well recognised, giving rise to its employment, as a preliminary measure, where the laryngoscope is about to be used. This local action is, according to Boehm and others, simply an abolished reflex without any change in the sensibility of the part; but against this view there is strong evidence. Gatumeau saw complete anæsthesia of the conjunctivæ induced by the bromide; and Bill observed the same phenomenon in the mucous membrane of the urinary tract. In spite of Voisin's opinion to the contrary, anæsthesia of the skin is, I venture to assert, one of the commonest symptoms to be met with in bromism. Professor J. M. Purser, of Dublin, has established the fact that the bromide impairs the cutaneous sensibility of frogs, whilst Guttman and Eulenberg have observed the same thing in rabbits.

The sensory impairment induced by the bromide in patients taking large doses of the salt is nearly always an analgesia, tactile or thermal impression being but very slightly interfered with. This loss of painful impressions is general, affecting the entire surface of the skin. The following case will serve to illustrate the point:—

J. B., aged 19, a worker in a hat manufactory, a robust and healthy-looking young woman, came under treatment for "cramps" of the right leg, and fits occurring during sleep. On the 18th of December, 1878, half a drachm of the bromide of potassium was administered thrice daily and continued until the 28th of January (following month), when the quantity was increased to 50 grains three times in the day. On February 3rd, it was noticed that she was exceedingly sleepy; the pupils dilated, though they reacted to light. Speech rather thick, but no amnesic aphasia; the tongue coated, and the breath fœtid. The superficial reflexes were completely abolished, whilst the so-called patella tendon reflex was increased on both sides. Tactile sensation was almost normal, but a pin could be inserted into the skin all over the body without any complaint of pain; indeed such a stimulus simply excited a tactile sensation.

In another case, that of a lad aged 14, who had suffered from epilepsy for several years, 40 grain doses of the bromide administered for three weeks induced a high degree of analgesia, so much so, that the case was selected as an example of toxic anæsthesia upon which to test the influence of *metals*. In this case,

also, there was a very marked increase in the knee phenomenon, and a diminution in the plantar and other superficial reflexes.

I might be allowed, in passing, to put on record my firm belief that the knee phenomenon is not a true reflex, but is simply an expression of the local condition of the quadriceps femoris muscle. I shall not enter upon a discussion of the point here, beyond laying stress upon the influence of bromide of potassium in exalting the so-called knee reflex, whilst it depresses the common skin reflexes; most pharmacologists being agreed that the salt lowers the spinal-reflex centres, whilst, on the other hand, Dr. Purser has shown that if a not too concentrated solution be applied directly to the muscles of a frog, a tetanic spasm is the result.

[TO BE CONTINUED.]

On the motion of Dr. BARRON, seconded by Dr. YELD, and carried unanimously, it was agreed that the paper which Prof. Lebour had kindly promised to read before the Society on "The geological distribution of goitre," should be taken at the March meeting, after the discussion on the prevalent diseases of the district.



CONTENTS.

ORDINARY MEETING.

PREVALENT DISEASES OF THE DISTRICT.

PATHOLOGICAL SPECIMENS.

EXHIBITION OF PATIENT.

ON PLEURITIC EFFUSIONS.

By CHARLES GIBSON, M.D.

NOTES OF A CASE OF OVARIOTOMY.

By T. W. BARRON, M.D.

ON ANÆSTHESIA (*Continued*).

By DAVID DRUMMOND, M.A., M.D.